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Association of Primary Renal Non-Hodgkin's Lymphoma With Mesangioproliferative Glomerulonephritis

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A 27-year-old male developed nonoliguric renal failure. Renal biopsy of the left kidney showed infiltration by a diffuse large-cell non-Hodgkin's lymphoma (NHL). Laparoscopy, CT scans of the abdomen and thorax, and bone-marrow biopsy revealed no further manifestations of lymphoma. Primary renal NHL was diagnosed. The patient attained complete remission with cyclophosphamide, adriamycin, vincristine, and prednisone (CHOP) chemotherapy and remained disease-free for 13 years. Eight years after his first presentation, the patient developed acute oliguric renal failure with nephrotic syndrome. Mesangioproliferative glomerulonephritis was diagnosed in a biopsy of the left kidney. Chronic hemodialysis was required until cadaver kidney transplantation was successfully performed 5 years later. Although the association of NHL and glumerulonephritis has been described several times before, to our knowledge this is the first report of glomerulonephritis in primary renal lymphoma. © 1996 Wiley-Liss, Inc.

Key words: primary renal lymphoma, mesangioproliferative glomerulonephritis, causal relationship

INTRODUCTION

Many authors have described lymphomatous infiltration of the kidney in patients with disseminated non-Hodgkin's lymphoma (NHL) [1–3]. Before the advent of effective chemotherapy, renal involvement was a common extranodal manifestation, secondary in incidence only to gastrointestinal and pulmonary manifestations. In consequence of early diagnosis and successful chemotherapeutic treatment of lymphoma, infiltration of the kidneys is observed less frequently nowadays.

Primary renal lymphoma (i.e., lymphoma without other extranodal or nodal manifestations) is a rare entity. Only about 40 cases have been reported [4,5]. One pathogenetic aspect may be the lack of lymphatic tissue in the kidneys.

The association of Hodgkin's lymphoma or NHL with glomerulonephritis has been recognized for decades [6]. Glomerulonephritis is thought to be a paraneoplastic manifestation of simultaneous or metachronic lymphoma [7]. Glomerulonephritis seems to be less frequent in NHL compared to Hodgkin's disease [7–9]. While minimal-change glomerulonephritis predominates in the mixed-cell type of Hodgkin's disease [8–10], several different types of glomerulonephritis are observed in NHL [7,8,11] (Table I).

We report on a patient who acquired mesangioproliferative glomerulonephritis 9 years after successful treatment of a primary renal NHL.

CASE REPORT

A 27-year-old Caucasian man was admitted to the hospital on December 19, 1981. He had complained of increasing abdominal discomfort, diffuse abdominal pain, and diarrhea for 6 weeks. He had experienced fatigue and malaise for a few months and had lost 4 kg body weight. Three days before admission, serum creatinine had been found to be 5 mg/dl. Physical examination of the pale patient did not show any lymphadenopathy. Blood

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TABLE I. Review of the Literature on NHL-Associated Glomerulonephritis*

Primary author and reference no.	Year	Age/sex	NHL type	Glomerulonephritis type	Nephrotic syndrome	Sequence[a]
Low-grade NHL						
Sagel [53]	1971	58/m	Lymphocytic	Crescentic	Yes	Simultaneous
Gagliano [7]	1976	65/m	Lymphocytic	Lipoid nephrosis	Yes	Simultaneous
Petzel [54]	1979	57/m	Lymphocytic	Crescentic	No	Simultaneous
		48/m	Lymphocytic	Crescentic	No	Simultaneous
Flury [65]	1982	65/f	Lymphocytic	Membranoproliferative	Yes	GN, 8 m, NHL
Herskowitz [72]	1982	7/?	Convoluted T-cell lymphocytic	Focal glomerulosclerosis	Yes	Simultaneous
Banks [56]	1984	51/m	Lymphocytic	Proliferative	Yes	GN, 18 m, NHL
Biava [55]	1984	57/m	Lymphocytic	Crescentic	No	?
Gonzalez [49]	1986	67/f	Mixed diffuse lymphocytic	Mesangioproliferative	Yes	Simultaneous
Jacquot [63]	1987	52/f	Small lymphocytic	Endocapillary proliferative	No	NHL, 9 y, GN
Pollock [74]	1988	48/m	Lymphocytic	Focal necrotizing	Yes	Simultaneous
Rosenmann [75]	1988	41/m	Diffuse small cleaved cell	Membranous	Yes	NHL, 18 m, GN
Schurgers [58]	1988	47/m	Lymphoplasmocytoid	Focal and segmental glomerulosclerosis	No	NHL, 6 y, GN
Cronin [41]	1990	70/m	Lymphocytic	Minimal-change	Yes	Simultaneous
Weber [43]	1990	46/m	Lymphocytic	Proliferative	No	Simultaneous
Poch [11]	1991	51/m	Small cleaved cell	Focal necrotizing	No	Simultaneous
Gärtner [45]	1991	n = 3	Immunocytoma (2 cases), unclassified (1 case)	Membranoproliferative	?	?
Rault [8]	1992	50/f	Small noncleaved cell	Mesangial hypercellular	Yes	Simultaneous
		68/m	Diffuse lymphocytic	Mesangial/endocapillary hypercellular	Yes	NHL, 17 m, GN
Rollino [47]	1992	59/m	Lymphocytic	Membranoproliferative	No	NHL, 11 m, GN
Chronic lymphatic leukemia						
Brodovsky [61]	1968	49/f	Chronic lymphatic leukemia	?	Yes	NHL, 4 y, GN
Kerkhoven [70]	1973	67/m	Chronic lymphatic leukemia	?	Yes	Simultaneous
Gilboa [62]	1979	62/m	Chronic lymphatic leukemia	Membranoproliferative	Yes	NHL, 6 y, GN
		53/f	Chronic lymphatic leukemia	Membranoproliferative	Yes	NHL, 8 y, GN
Frehally [60]	1981	64/m	Chronic lymphatic leukemia	Membranoproliferative	Yes	Simultaneous
Strippoli [73]	1982	65/m	Chronic lymphatic leukemia	Mesangioproliferative	Yes	GN, 18 m, NHL
Dabbs [52]	1986	n = 3	Chronic lymphatic leukemia	Minimal-change	?	?
			Chronic lymphatic leukemia	Minimal-change	?	?
			Chronic lymphatic leukemia	Membranoproliferative	?	?
Seney [59]	1986	50/f	Chronic lymphatic leukemia	Minimal-change	Yes	NHL, 1 y, GN
		55/m	Chronic lymphatic leukemia	Membranoproliferative, type II	Yes	NHL, 8 y, GN
		71/m	Chronic lymphatic leukemia	Membranoproliferative, type I	Yes	NHL, 2 y, GN
		60/m	Chronic lymphatic leukemia	Minimal-change	Yes	NHL, 17 y, GN
Gärtner [45]	1991	n = 4	Chronic lymphatic leukemia	Membranoproliferative	?	?
Moulin [46]	1991	n = 13	Chronic lymphatic leukemia (11 cases)	Membranoproliferative (8 cases)	Yes (9 cases)	Simultaneous (7 cases)
		(8/m, 5/f)	lymphocytic (2 cases)	Focal segment (1 case), crescentic (1 case), advanced sklerosing (1 case), membranous (1 case), mesangial hypertrophy (1 case)		
Cutaneous lymphoma						
Belghiti [64]	1981	16/f	T-cell	Focal glomerulosclerosis	Yes	Simultaneous
Ramirez [57]	1981	70/m	Mycosis fungoides	IgA nephritis	No	Simultaneous
		56/m	Mycosis fungoides	IgA nephritis	No	NHL, 3 y, GN
Allon [42]	1988	53/m	Mycosis fungoides	Minimal-change	Yes	NHL, 17 m, GN
Torrelo [76]	1990	49/f	Mycosis fungoides	Immunotactoid	Yes	NHL, 5 y, GN
Moe [44]	1993	66/f	Mycosis fungoides	IgA nephritis	No	NHL, 6 m, GN
		76/m	Mycosis fungoides	Membranous	Yes	Simultaneous
		44/m	Mycosis fungoides	Focal immune complex	No	NHL, 1 y, GN

(Continued)

pressure was 105/80 mm Hg, pulse was 104 bpm, the lungs were clear, and heart sounds were normal. Abdominal ultrasonography showed enlarged kidneys (right, 12.5 × 7.7 cm; left, 12.4 × 7.1 cm) with homogeneous structure but no evidence of urinary obstruction or retroperitoneal adenopathy. The spleen was not enlarged

TABLE I. (Continued)

Primary author and reference no.	Year	Age/sex	NHL type	Glomerulonephritis type	Nephrotic syndrome	Sequence[a]
High-grade NHL						
Ghosh [68]	1970	74/m	Lymphosarcoma	?	Yes	GN, 11 m, NHL
Muggia [69]	1971	36/f	Reticulum cell-type	Proliferative/membranous	?	Simultaneous
		60/f	Reticulum cell-type	?	Yes	Simultaneous
Hyman [48]	1973	11/m	Burkitt's lymphoma	Membranoproliferative	Yes	NHL, 4 m, GN
Rabkin [71]	1973	62/m	Lymphosarcoma	Membranous	Yes	GN, 8 m, NHL
Matsui [50]	1990	60/m	Diffuse large-cell	Mesangium proliferative	Yes	Simultaneous
Weinstein [9]	1990	55/f	Diffuse large-cell	Crescentic	No	GN, 13 m, NHL
Munker [51]	1991	49/m	Lennert's lymphoma	Mesangioproliferative	Yes	Simultaneous

*Survey of 68 cases of NHL-associated glomerulonephritis (GN). Male sex prevails (m = 39, f = 18, sex unknown = 11). In most cases, simultaneous appearance of NHL and GN was observed (n = 26). In 18 cases, NHL preceded GN. Only six times was GN followed by NHL. High-grade NHLs are outnumbered by lymphocytic lymphoma and chronic lymphatic leukemia. Membranoproliferative GN was the most frequent type of NHL-associated GN. Forty of the 68 cases of GN were associated with nephrotic syndrome.

[a]y, year; m, month.

(8.5 × 4.5 cm). CT scans of the abdomen confirmed the presence of enlarged kidneys without additional pathologic findings.

On December 29, 1981, laparoscopic biopsy of the left kidney was performed under general anesthesia. Histologic investigation revealed diffuse interstitial infiltration of the parenchyma by a centroblastic lymphoma of polymorphic subtype, corresponding to a diffuse large-cell lymphoma, which was a noncleaved variant according to the Working Formulation (Fig. 1a). Further staging procedures included chest X-ray, CT scan of the thorax, pedal lymphangiography, bone scintigraphy, exploratory laparotomy, and liver biopsy, but none showed evidence of NHL. A bone-marrow film and unilateral biopsy specimen from the iliac crest did not reveal any signs of lymphoma. Rhinoscopy and laryngoscopy revealed no abnormalities.

The patient had normochromic, normocytic anemia. Hemoglobin concentration was 11.1 g/dl. The leukocyte count was 7,000 × 10^9/l, with the differential count showing 84% polymorphs, 3% bands, 1% basophils, 10% lymphocytes, and 2% monocytes. Platelets were 226,000 × 10^9/l. His creatinine and blood urea nitrogen (BUN) were elevated to 6.7 mg/dl and 78 mg/dl, respectively. The erythrocyte sedimentation rate was 32/75 mm, uric acid was 9.1 mg/dl, and alkaline phosphatase was 215 u/l. Blood gas analysis revealed metabolic acidosis with respiratory compensation. Serum calcium, phosphorus, potassium, sodium, haptoglobin, ferritin, serum bilirubin, aspartate-amino transferase (AST), alanine-amino transferase (ALT), cholinesterase, antistreptolysin titre (ASL), and tests for antibodies to glomerular basement membrane antibodies C3c, C3, and C4 complement were within normal limits. Immunoelectrophoresis of serum and urine provided no evidence of paraprotein.

Urinary output ranged from 1.3 l–2.6 l per 24 hr. Urinalysis showed 0–1 red blood cells/high power field (HPF), 1–4 white cells/HPF, and amorphic salts. No hyaline casts were visible. Urinary protein concentration was 0.1 g/l. Urine culture and cytology were negative.

On January 16, 1982, CHOP (cyclophosphamide, adriamycin, vincristine, and prednisone) chemotherapy was initiated, with the dose of cyclophosphamide and adriamycin adapted to impaired renal function. Creatinine and BUN fell from peak values of 9.8 mg/dl and 120 mg/dl, respectively, to 2.9 mg/dl and 46 mg/dl over 1 month. Until August 1982, seven further courses of CHOP were applied, and creatinine and BUN decreased to 2.4 mg/dl and 37 mg/dl, respectively. During restaging after six courses in May 1982, the shape and size of the kidneys were normal on ultrasonography and computed tomography (right, 10 × 5 cm; left, 10.2 × 5.2 cm).

Percutaneous rebiopsy of the left kidney showed normal renal tissue. There was no evidence of lymphoma or glomerulonephritis. Renal ^{131}I-hippurate scintigraphy revealed markedly reduced function of the right kidney (right, 27%; left, 73%). During annual follow-ups, the patient remained in remission without further treatment. Creatinine values ranged from 1.9–2.1 mg/dl, and BUN from 29–34 mg/dl.

On December 14, 1989, eight years after the diagnosis of high-grade lymphoma, the patient was readmitted to our hospital. He complained of increasing dyspnea and epistaxis during the last 2–3 weeks. He had lost 5 kg body weight during the last 6 weeks. The patient was pale, his blood pressure was 250/150 mm Hg, his pulse was 108 bpm, and he was tachypneic. Physical examination showed no further abnormalities.

Clinical test values were hemoglobin 8.8 g/dl, leukocytes 12.700 × 10^9/l, and thrombocytes 201,000 × 10^9/l. Creatinine clearance was 46 ml/min early in the course of the disease. Peak values for creatinine and BUN were

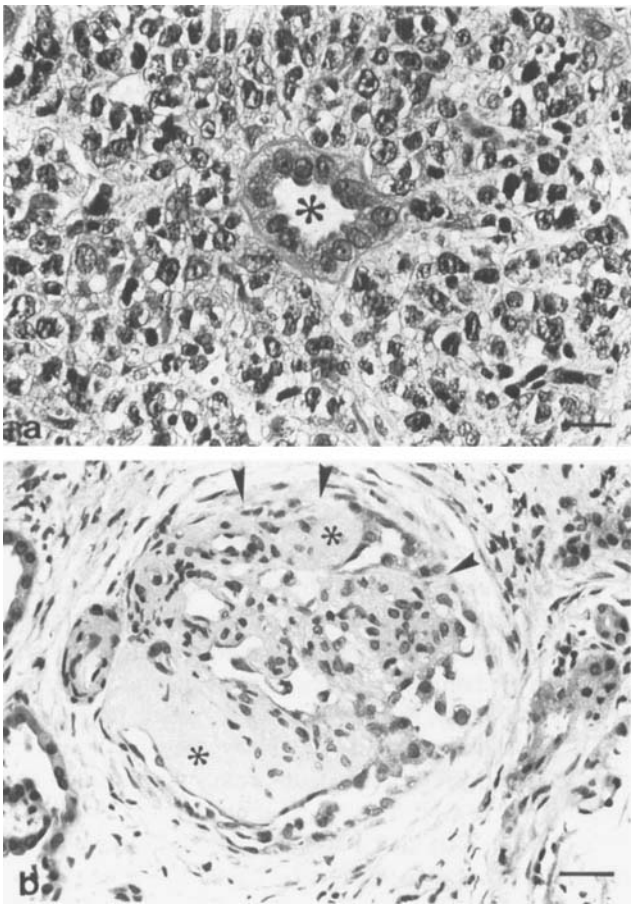

Fig. 1. a: Biopsy of left kidney. Diffuse infiltration of renal interstitium by a centroblastic lymphoma. There is a polymorphic tumor-cell population with many centroblasts around a renal tubule (asterisk). H&E staining. Bar, 40 μm. b: Biopsy of left kidney 8 years later. Sclerosing stage of mesangioproliferative glomerulonephritis with panmesangial involvement and capsular synechiae (arrowheads), as well as pronounced matrix (asterisks) and moderately increased cellularity. H&E staining. Bar, 80 μm.

9.1 mg/dl and 124 mg/dl, respectively. Besides an erythrocyte sedimentation rate of 115 mm in the first hour, C-reactive protein of 14.7 mg/dl, and lactate dehydrogenase (LDH) of 354 U/l, all other blood tests revealed normal results. Urinalysis showed 5–15 erythrocytes/HPF; urine was positive for protein and erythrocytes. Abdominal ultrasonography and CT scans provided no evidence of recurrent lymphoma. The kidneys were small (right, 7.6 × 4.0 cm; left, 8.9 × 3.6 cm) and showed echogenic parenchymal changes. Percutaneous biopsy of the left kidney was performed to evaluate renal impairment. Histologic examination showed mesangioproliferative glomerulonephritis (Fig. 1b). After renal biopsy, massive intraabdominal bleeding occurred, and packed red blood cells had to be transfused.

On December 16, 1989, hemodialysis was started, using an arteriovenous fistula which had been created eight years ago. The patient had required chronic dialysis for 5 years. He finally received a currently well-functioning cadaver kidney transplant in March 1995.

DISCUSSION

The first description of kidney involvement in 9 patients with lymphoma was presented in 1878 by Sutton and Turner [12]. Renal involvement was a frequent extranodal manifestation of Hodgkin's lymphoma and NHL before the development of aggressive chemotherapy. It was often found at autopsy in disseminated disease, equivalent to stage IV according to the Ann Arbor classification [2,13–18]. A retrospective pathological analysis of 696 cases of Hodgkin's disease and NHL from 1916–1962 demonstrated lymphomatous infiltration in 33.5% of all cases. Kidney infiltration was bilateral in 74% and unilateral in 26% [2]. In NHL with bone-marrow infiltration, the incidence of renal involvement was 63%, compared to 39% in NHL without bone-marrow infiltration.

The recent era of aggressive chemotherapeutic treatment of NHL was accompanied by a decline in renal involvement [1,3,19]. In clinical studies, renal involvement is seldom observed now. Its prevalence ranges from 2.4–14% [20,21]. Typical manifestations are parenchymal kidney infiltration and perirenal extension with vascular and ureteral obstruction [1,3]. Intraluminal tumorous thrombi are rare [22]. Ultrasonography and CT scans of the kidneys most often show multiple lesions (54%) or diffuse infiltration (36%), but solitary lesions (10%) may also occur [3]. Only a minority of patients with renal involvement present with impaired renal function or renal failure [3,16,23–26]. Acute renal failure was seldom the initial manifestation of disseminated NHL. In 17 cases described up to 1986 with renal failure as initial symptom, histologic examination often revealed bilateral diffuse lymphoma with massive enlargement of the kidneys [14,16,21,26,27]. Survival was poor, with most patients dying within 9 months of diagnosis [26]. Children with Burkitt's lymphoma frequently seem to suffer from renal involvement [28]. In most cases, however, renal failure is not a consequence of renal infiltration but of retroperitoneal lymphoma obstructing the ureters. Hypercalcemia, hyperuricemia, and renal amyloidosis may be further causes of renal failure in patients with lymphoma [11,13,26,29]. In general, renal function rapidly improves after chemotherapy or radiotherapy [13,26]. Renal involvement could be accepted as the leading cause of death in only 0.5% of 696 patients with lymphoma [2].

While disseminated lymphoma frequently involves the kidneys, primary lymphoma of one or both kidneys without any other extranodal or nodal involvement is very rare [4,30–33]. Due to the lack of lymphatic tissue in the

kidney, the existence of primary renal lymphoma has been questioned, and the perirenal tissue was thought to be the origin of these renal lymphomas [5,14,18,25,32,34]. Another hypothesis maintains that a reactive lymphatic population homes to the kidney, and that lymphoma develops after an inflammatory process [4,23]. A subgroup of renal lymphomas may be explained by the MALT concept (mucosa-associated lymphoid tissue) developed by Isaacson and Wright [6] and Parveen et al. [35]. Even in cases referred to a "primary renal" lymphoma, lymphadenopathy or mediastinal enlargements have been described [13,25]. Therefore, Malbrain et al. [32] specified five criteria for primary renal lymphoma, including: 1) renal failure at initial presentation, 2) bilateral enlargement of the kidneys without obstruction and other organ or nodal involvement, 3) diagnosis made by renal biopsy, 4) absence of other causes of renal failure, and 5) rapid improvement of renal function after chemotherapy or radiation. According to these criteria, 9 adult patients with primary renal lymphoma have been described from 1980–1994, five of whom died within 1 year [32]. Unilateral renal lymphoma, which is a rarely-reported distinct entity, does not cause renal failure and may be cured by surgical kidney resection [5,30,32,36–38]. Primary lymphoma of the kidney may develop in patients with AIDS [38,39]. Brouland et al. [4] recently reported on 36 cases of primary unilateral and bilateral renal lymphoma. To summarize, it must be emphasized that there is no satisfactory clinical definition of "primary renal" lymphoma; even the most consistent proposal by Malbrain et al. [32] remains arbitrary and would not correctly classify primary renal lymphoma after dissemination.

Our patient initially presented with renal failure and enlarged kidneys in ultrasonography and CT scans. Thorough diagnostic procedures revealed no other manifestation of lymphoma and no other cause of renal failure. Diagnosis was made by renal biopsy, and renal function dramatically improved after chemotherapy. Thus, this case clearly meets the criteria of Malbrain et al. [32] of "primary renal" lymphoma.

The coincidence of glomerulonephritis with nephrotic syndrome and neoplasia has attracted particular attention for 30 years [40]. About 10% of all patients with glomerulonephritis are estimated to have malignant diseases. The association of localized or disseminated NHL with simultaneous minimal change [41,42], membranous [43,44], membranoproliferative [45–48], mesangioproliferative [49–51], crescentic [9,52–55], focal-necrotizing [56], and IgA glomerulonephritis [44,57] has been described. Table I presents a survey of the literature reporting NHL associated with glomerulonephritis. In all these cases except one [58], concomitant renal involvement of lymphoma was ruled out by renal biopsy. In contrast to Hodgkin's disease, in which minimal-change glomerulonephritis predominates, histologic patterns of glomerulonephritis in NHL are hetereogenous. B-cell NHLs are associated with many different kinds of glomerulonephritis, while cutaneous T-cell lymphoma tends to coexist with IgA nephropathy [44]. Glomerulonephritis is more frequent in low-grade B-cell NHL [49,52], especially in chronic lymphatic leukemia [8,46,54,59–62], than in intermediate and high-grade B-cell NHL [9,50]. As in our case, the association of glomerulonephritis and NHL is mostly seen in males [11,49]. In some instances, additional cryoglobulinemia with generalized vasculitis has been diagnosed [8,43,46,47,62,63]. Renal function is usually not altered in minimal-change glomerulonephritis associated with Hodgkin's disease [8,10]. In contrast, the more severe types of NHL-associated glomerulonephritis frequently result in full-blown nephrotic syndrome [7,8,48,50, 53,56,63–65], impaired renal function [7–9,11,46], or renal failure [41,47,49,50].

Whereas renal failure in idiopathic nephrotic syndrome rarely responds to treatment, renal function in lymphoma-associated glomerulonephritis generally improves after initiation of chemotherapy and may even return to normal [8,11,41,43,46,49,50,60]. However, renal function in NHL-associated glomerulonephritis may be normal at time of diagnosis [8] and deteriorate subsequent to chemotherapy [11,51].

In general, NHL and associated glomerulonephritis are manifested simultaneously [9]. Under these conditions, glomerulonephritis is usually referred to as a paraneoplastic syndrome [7,8,11,46,49]. Glomerulonephritis preceded NHL in only 6 cases [9,56,65,68,71,73] or, as in our patient, developed months or years [8,42,44,47, 48,57–59,61–63,75,76] after onset of lymphoma.

In our case, the lag time between diagnosis of renal lymphoma and the development of mesangioproliferative glomerulonephritis was 8 years. When glomerulonephritis was found in 1989, the patient had been in complete remission for 7 years, and renal biopsy showed no signs of relapsed lymphoma. Furthermore, the patient remained in stable remission for another 6 years, when cadaver kidney transplantation was finally performed successfully in 1995. Thus, the patient had been in complete remission for 7 years and remained in remission for at least 6 additional years. It might be argued that in our case the phenomenon of glomerulonephritis associated with NHL was purely coincidental rather then paraneoplastic [48,56]. Nevertheless, the manifestation of two different entities in the same organ raises the question of the interdependence of NHL and glomerulonephritis. Further cases of metachronic glomerulonephritis have been described [56,59,61–63,73,76].

The pathogenesis of NHL-associated glomerulonephritis is not yet understood. An immunological link between the pathogenesis of lymphoma and glomerulonephritis has been postulated. When immunoglobulins produced

by B-cell lymphoma were found in renal biopsies, they were thought to be responsible for glomerular damage [43,46,62]. This concept was placed in question by observations of glomerulonephritis associated with T-cell lymphoma [44,49,51,64]. A current pathogenetic concept of paraneoplastic glomerulonephritis focuses on tumor antigens within immune complexes, which are deposited on the glomerular basement membrane [7,49,53]. Animal studies provided further evidence for this pathogenetic mechanism [66,67]. However, metachronic glomerulonephritis as found in our patient remains enigmatic.

In summary, we present a unique case of primary renal NHL, associated with metachronic mesangioproliferative glomerulonephritis. Although a thorough review of the literature was performed, further cases of glomerulonephritis in primary renal lymphoma could not be identified. The occurence of the two diseases in the same patient deserves special attention, because there may be a causal relationship between NHL and glomerulonephritis.

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